Substances that influence the epiphyseal plate

- Sex Hormones
  - Androgens and estrogen stimulate bone formation
    - Accelerate Closure of epiphyseal plate
    - Puberty causes closure of majjor long bones 2 years earlier in females vs. males
      - Females are shorter
- Growth Hormone (Somatotropin)
  - Secreted by anterior pituitary gland--> stimulates bone formation
    - Excess GH (child) → Gigantism
    - Deficiecy GH (child) → Piituitary Dwarfism
    - Excess GH (adult) → Acromegaly
      - Thickening of facial bones and enlargement of hands and feet
- Vitamin A
  - Acts to regulate bone degradation and deposition (remodeling)
    - Excess Vitamin A → More rapid erosion of cartilage--> permanent closure epiphyseal plate --> Dwarfism
    - Deficiecy Vitamin A → Decrease in growth rate, lack of bone resorption, fail to enlarge critical bones fast enough
- Vitamin D
  - Controls normal absorption of Ca2+ from the gut
    - Deficiecy Vitamin D (child) → Rickets
      - Lack of Ca2+ leads to no mineralization of cartilage in epiphyseal plate, Bowed bones
    - Deficiecy Vitamin D (adult) → Osteomalacia
      - Lack of Ca2+, during remodeling, newly formed osteos do no calcify sufficiently, bones are weakened and break easily
- Vitamin C
  - Cofactor for the hydroxylation of proline in alpha chains of forming procollagen
    - Lack of hydrogen bonding btwn hydroxy proline = weak tropocollagen → Scurvy
      - Collagen in bone matrix is weak and strong osteoid is not fromed --> thinner cortical bone --> weak bones, growth and repair of fractures retarded
- Age related bone loss
  - Loss of bone from all parts of skeleton with aging
    - Rate of bone deposition decreases (resorption stays the same)
    - Thinning of cortical bone, loss of trabecula throughout bone mass
    - Severe Bone Loss → Osteoporosis
      - Severe bone loss of bone mass with aging, cannot maintain mechanical support of body weight, afflicts 25% of all postmenopausal women, fractures, backaches,